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Galactose treatment in focal segmental glomerulosclerosis

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To the editors

We read with great interest the recent report by Sgambat et al [1] describing the use of galactose as antiproteinuric therapy in 7 children with steroid-resistant nephrotic syndrome. Serum P_{alb} activity of each patient was reduced after 16 weeks of therapy. However, based on the lack of a decline in proteinuria, the authors conclude that galactose treatment is ineffective and they recommend that galactose should not be pursued as a treatment option in patients with FSGS in native kidneys. They suggest that its use in children with recurrent FSGS post-transplantation may be considered. We propose that the negative findings in this case series do not warrant such a sweeping conclusion. The sample size is small and includes heterogeneous patients with MCNS, FSGS in their native kidneys and recurrent disease in allografts. The duration of disease is long (duration 28±23 months) and galactose may have been started after there had been permanent, irreversible glomerular damage. There is ample *in vitro* evidence that galactose can prevent the permeability effects of FSGS serum and there are several case reports demonstrating the efficacy of oral galactose supplementation in adult patients with FSGS in their native kidneys and in renal transplants [2,3]. Preliminary data from the FONT trial (NCT00814255) suggest that some patients treated with oral galactose achieve a significant reduction in proteinuria in parallel with a decline in P_{alb} during 24 weeks of therapy (unpublished observations). In the light of these findings, novel biomarkers may be needed to identify patient subgroups who are likely to respond to galactose treatment and a longer period of therapy may be required to fully realize the benefit of therapy. Agents such as rituximab, with response rates of approximately 20% in pilot studies of steroid resistant nephrotic syndrome are still under active investigation. Since galactose is non-toxic and since other proven therapeutic options are limited, it may be premature to abandon investigation of galactose and other agents that may inhibit the activity of circulating permeability factors. Additional trials in larger samples of well characterized patients are warranted and prolonged therapy may show that galactose will reduce proteinuria in at least some patients.

References

1. Sgambat K, Banks M, Moudgil. effect of galactose on glomerular permeability and proteinuria in steroid-resistant nephrotic syndrome. Pediatr Nephrol. 2013; 28:2131–2135. [PubMed: 23793883]
2. Kopač M, Meglič A, Rus RR. Partial remission of resistant nephrotic syndrome after oral galactose therapy. Ther Apher Dial. 2011; 15:269–272. [PubMed: 21624074]
3. Jhaveri KD, Naber TH, Wang X, Molmenti E, Bhaskaran M, Boctor FN, Trachtman H. Treatment of recurrent focal segmental glomerular sclerosis posttransplant with a multimodal approach including high-galactose diet and oral galactose supplementation. Transplantation. 2011; 91:e35–36. [PubMed: 21383598]

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